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Original Article

The long-term effect of exercise on vascular risk factors and aerobic fitness in those with transient ischaemic attack: a randomized controlled trial

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Objective: Exercise has beneficial effects on vascular risk factors in transient ischaemic attack (TIA) patients within the sub-acute phase. This study examined whether TIA patients randomized to an early exercise and education programme within 2 weeks of TIA diagnosis would demonstrate improvements in cardiovascular risk factors and aerobic fitness 12 months postdiagnosis compared with control patients.

Methods: A single-centre, randomised, parallel-group clinical trial. Sixty TIA patients (69 ± 11 years) completed a vascular risk stratification baseline assessment and a physical fitness examination. Individuals were randomized to either an 8-week early exercise and education or control group. Fifty-one patients attended postintervention assessments that were completed immediately (postintervention) and 12 months after (12PI).

Results: A significantly greater improvement in resting SBP was observed between baseline and postintervention for EX than for CON (−11 mmHg cf. −1 mmHg, respectively; $P < 0.05$). The improvement in SBP was maintained between postintervention and 12PI ($P > 0.05$). Similar findings were demonstrated for BMI, bodyweight and peak oxygen uptake ($P < 0.05$). Exercise blood pressure, pulse pressure and double product (SBP x heart rate; an indication of myocardial workload) were significantly lower at postintervention and 12PI for EX than for CON (all $P < 0.05$).

Conclusion: An 8-week exercise programme soon after TIA resulted in beneficial changes in resting and exercise blood pressure that were maintained for 12 months.

Clinical trial registration: http://www.anzctr.org.au/ Trial Registration Number:ACTRN12611000630910

Keywords: long-term follow-up, physical exercise, SBP, transient ischaemic attack

Abbreviations: CAD, Coronary artery disease; CON, Control group; CVD, Cardiovascular disease; RPE, Ratings of perceived exertion; TIA, Transient ischaemic attack; VO_2peak, peak oxygen consumption; 12PI, 12-month postintervention

INTRODUCTION

Stroke is a leading cause of death and adult disability worldwide [1]. In the United States, it is estimated that of the nearly 795 000 people who experience a stroke, 23% (~185,000) are recurrent events [1], although nearly 15% of all strokes are heralded by a transient ischaemic attack (TIA) [2]. A TIA is a transient episode of neurological dysfunction caused by focal brain, spinal cord or retinal ischemia, without acute infarction [3]. Following TIA, the short-term risk of stroke is between 3 and 10% at 2 days and between 9 and 17% at 90 days [4,5], although nearly 12% of patients die within 1 year of initial TIA diagnosis [6]. To date, the primary preventive regime used to reduce the risk of a recurrent stroke or TIA is the prescription of antiplatelet agents, anticoagulants, blood pressure lowering and cholesterol-lowering treatments [7].

Exercise-based cardiac rehabilitation is an accepted component of the multifactorial secondary preventive strategy used to improve modifiable risk factors among coronary artery disease (CAD) patients [7–9]. As patients diagnosed with TIA share many of the predisposing modifiable vascular risk factors as CAD patients [10], an analogous secondary prevention strategy may be applicable for use with this population. Research has demonstrated the short-term efficacy of utilizing physical activity (aerobic and resistance exercise) as a secondary prevention strategy with nonacute TIA and ischaemic stroke patients, with improvements in SBP (~3–10 mmHg), DBP (~3 mmHg) and total cholesterol (~0.1–0.6 mmol/l) typically reported [9,11,12]. However, the long-term benefits of these interventions remain unknown despite the potential impact such information could make to chronic disease management [13].

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The purpose of this study was to examine whether TIA patients randomized to an early exercise and education programme would demonstrate improvements in cardiovascular disease risk profile (i.e. SBP) and aerobic fitness, and whether any benefit would be sustainable to 12 months.

MATERIALS AND METHODS

Participants

TIA diagnosis was based upon criteria from New Zealand's assessment and management guidelines [14]. To determine diagnosis and risk of further vascular events, the following clinical assessments and investigations were undertaken at the hospital: brain imaging [MRI or computed tomographic (CT) scan], $ABCD_2$ tool, routine blood tests (full blood count, electrolytes, glucose, lipids, creatinine and so on), carotid imaging, echocardiogram and electrocardiogram. Participants were eligible if they had been diagnosed with their first TIA, if they lived within the local district health board and if they did not meet exclusion criteria. Exclusion criteria were unstable cardiac conditions, uncontrolled diabetes mellitus, severe claudication, oxygen dependence, significant dementia, inability to communicate in English or unable to take part in exercise. Participants complied with drug treatment and standardized therapy in accordance with stroke physician recommendations.

Sample size

On the basis of the findings of Santos-Hiss *et al.* [15], and when using a two-sided 5% significance level and a power of 80%, a sample size of 30 patients per group ($n = 60$) was calculated to detect a reduction of 7 mmHg (pooled SD: 5.8 mmHg) in SBP between groups. This calculation anticipated a 25% drop-out rate.

Ethical approval

The trial was approved by New Zealand's Central Regional Health and Disabilities Ethics Committee and registered with the Australian and New Zealand Clinical Trials Registry (Trial Registration Number: ACTRN12611000630910). Written informed consent was obtained prior to participation.

Study design

The study was a single-centre, randomized, parallel-group design. TIA was confirmed by a specialist physician (G.M.) at Wellington Hospital within 7 days of symptom onset. Wellington hospital receives all TIA referrals from the local district health board. Eligible participants were invited to attend a baseline assessment at an exercise physiology laboratory within 2 weeks of TIA onset. Baseline included a health history questionnaire, a vascular risk assessment, a symptom limited exercise-ECG stress test and a two-stage exercise test on a cycle ergometer. The cycle test was used to assess aerobic fitness as well as cardiovascular changes that occurred during exercise (Table 1). Identical assessments were completed postintervention and at 12-month follow-up (12PI; Table 1). Further information on the study design can be found elsewhere [11].

Primary and secondary outcome measures

The primary outcome measure was resting SBP. Secondary outcome measures included other vascular risk factors (blood pressure, total cholesterol, high-density lipoproteins, total cholesterol: high-density lipoprotein ratio, fasted blood glucose, BMI, waist circumference, hip circumference, waist to hip ratio) and the cardiovascular and cardiorespiratory responses obtained during (i.e. heart rate, blood pressure) or following (i.e. estimated peak oxygen

TABLE 1. Study procedures for the assessment sessions and intervention

Baseline (BL) assessment	8-week intervention		PI and 12PI follow-up assessments
	EX	CON	
Health History Questionnaire [21] Vascular risk stratification [21] • Finger prick blood sample to assess total cholesterol, high-density lipoproteins; total cholesterol: high density lipoproteins ratio[a] and fasting blood glucose[b] • Seated/standing/supine blood pressure[c] • Smoking history • Family history of CVD • Waist and hip girth • Height, weight, body mass index Exercise ECG stress test Symptom limited exercise ECG treadmill stress test (modified Bruce protocol) to determine clinical suitability for participants to engage in the EX programme[d] Fitness assessment Participants completed a two stage exercise test on a cycle ergometer[e], equivalent to a power output of 30 W and 60 W, respectively. Each stage was 3-minutes in length. Blood pressure, heart rate and the ratings of perceived exertion (RPE) [22] were monitored in the final minute of both exercise stages. Participants maintained a cadence of 50 rpm throughout the test. Based on the data recorded, the following measures were reported: Heart rate, systolic and diastolic blood pressure, pulse pressure and double product. Submaximal estimates of oxygen consumption were elicited from the two exercise stages. Linear regression analysis, using the heart rate and oxygen uptake values from the two exercise stages, enabled a prediction in peak oxygen consumption to be obtained [21]	Exercise 2 x 90 minute exercise sessions per week *15 min walking & 15 min cycling* • Blood pressure, heart rate and RPE were measured prior to, during and following each bout of aerobic exercise • Participants exercised between 50 and 85% of maximal heart rate • Exercise intensity was increased by ~5% each week *Resistance exercise* • 60 min of resistance training, core-stability and postural exercises. • 30 min completed after walking, and 30 min on completion of cycling. Education • 1x30 min didactic group discussion each week to facilitate patients with a greater sense of understanding and management of their condition. The education programme was designed so that practitioners could teach, discuss and reinforce behaviours which are known to facilitate healthy behavior change [23]. • Constructed in line with the health belief model for behaviour change. • Focused on vascular risk factors, stroke prevention, nutrition, BP, adherence to medication, stress management and emotional and behavioural changes after TIA.	Educational information provided by Hospital Monthly telephone calls	See baseline assessment

[a]CardioChek, Hannover, Germany.
[b]Optium, Abbott Diabetes Care, Victoria, Australia.
[c]Stethoscope and Shygomomanometer, Accoson Works, London, England.
[d]Schiller, Baar, Switzerland
[e]Monark Ergometer, Sweden.

uptake) the cycle test (see Table 1). Pulse pressure (SBP – DBP) and double product (SBP x heart rate) were calculated after the exercise test.

Randomization

On completion of baseline, participants were randomized to an 8-week exercise and education intervention or to a usual care control group using computerized random numbers [11]. Randomization was undertaken by an investigator with no clinical involvement in the trial. Outcome assessors and data analysts were blinded to the allocation.

Exercise and education programme

Exercise and education intervention comprised two 90-min exercise sessions and one 30-min education session per week for 8 weeks. The control group received standard secondary prevention and educational information from the hospital (Table 1).

Statistical analyses

Pearson Chi-squared tests and independent-samples *t*-tests compared baseline descriptive statistics between exercise and education intervention and control participants. An independent-samples *t*-test was used to assess recurrent TIA between groups.

Repeated measures analysis of variance (ANOVA) was used to determine whether the condition that participants were randomised to (i.e. exercise and education intervention or control) influenced the outcome measures assessed (i.e. vascular risk factors, cardiovascular and cardiorespiratory measures obtained from the cycle test) at the different assessment sessions (baseline, postintervention, 12PI). Where significant differences were observed, Tukeys HSD post hoc test was used to identify where the differences were located. Partial eta squared (η_p^2) was used to demonstrate the strength of the effect of exercise (exercise and education intervention) on the various outcome measures with 0.0099, 0.0588 and 0.1379 representing a small, medium and large effect, respectively. Partial eta squared was calculated using the following formula:

$$\eta_p^2 = \frac{SS_{Effect}}{SS_{Effect} + SS_{Error}}$$

whereby SS_{Effect} is the estimated variance for a given outcome measure, and SS_{Error} is the error variance that is attributable to the effect. Data were analysed using the statistical package SPSS for Windows, version 20.

RESULTS

Participant recruitment, adherence and recurrent transient ischaemic attack risk

Recruitment took place between February and November 2011. Follow-up assessments were completed between May 2011 and January 2013. Of the 60 participants who completed baseline, 51 attended the 12PI (Fig. 1). Four individuals at postintervention (8%) revealed their treatment allocation to the outcome assessors. There were no differences in individual characteristics or medication use at baseline between groups ($P > 0.05$; Table 2). Between the baseline and the 12PI assessment, two exercise and education intervention and four controls experienced recurrent TIA ($P > 0.05$). Mean medication use at postintervention and 12PI was similar to baseline for both exercise and education intervention (2.89 ± 1.03 and 2.81 ± 1.06 for postintervention and 12PI, respectively) and control (2.84 ± 1.03 and 2.79 ± 1.05 for postintervention and 12PI, respectively) ($P > 0.05$).

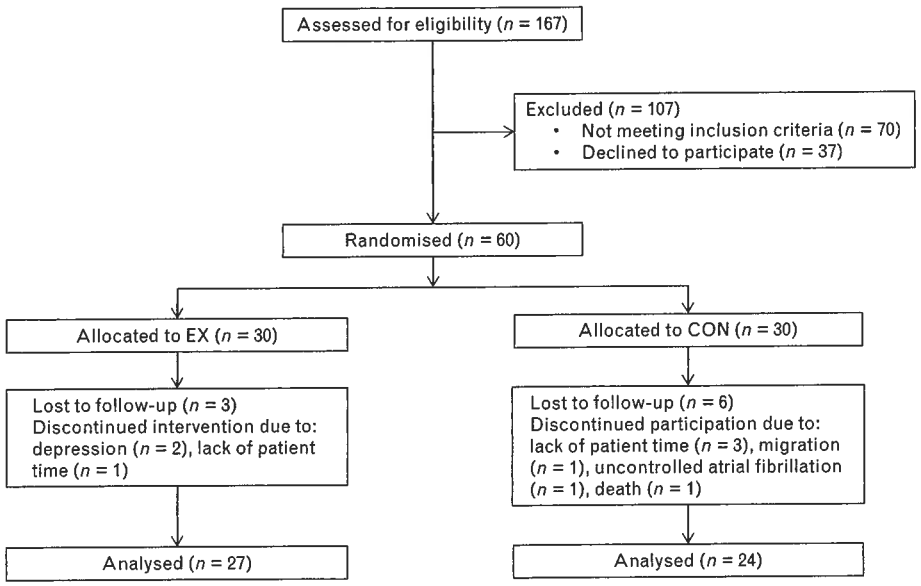

FIGURE 1 Participant recruitment.

TABLE 2. Baseline descriptives for exercise and education intervention and control participants who completed all three assessments (baseline, postintervention, 12PI)

		EX		CON		
		n	%	*n*	%	*P*
Participants (*n*)		27	53	24	47	
Age (years)		65 ± 11		69 ± 10		0.218
Sex (*n*)	Male	15	56	11	46	0.498
	Female	12	44	13	54	
Descent (*n*)	European	24	89	21	89	0.761
	Maori	1	4	0	0	
	Pacifika	1	4	1	4	
	Asian	1	4	1	4	
	Indian	0	0	1	4	
Family history of CVD	Myocardial infarction	14	52	13	54	0.923
	Heart surgery	4	15	3	13	0.564
	Stent	1	4	3	13	0.094
	Catheter	0	0	3	13	0.083
	Heart defect	3	10	1	4	0.210
	Stroke	11	41	11	46	0.721
Personal history of CVD	Hypertension	17	63	17	71	0.561
	Clinical diagnosis of hypertension (≥140/90 mmHg)	11	47	7	29	0.398
	High cholesterol	14	52	13	54	0.872
	Diabetes	3	10	5	21	0.351
	Heart problems	4	15	9	38	0.071
	Artery diseases	2	7	2	8	0.905
	Thyroid disease	0	0	1	4	0.293
	Lung disease	2	7	2	8	0.905
	Asthma	6	22	5	21	0.907
	Cancer	5	19	6	25	0.583
	Kidney disease	2	7	1	4	0.632
	Hepatitis	2	7	1	4	0.632
Signs and symptoms of CVD	Chest pain	9	33	9	38	0.762
	Dyspnea	17	63	15	63	0.973
	Heart palpitations	9	33	8	33	1.000
	Skipped heart beats	9	33	5	21	0.328
	Heart murmur	1	4	4	17	0.125
	Intermittent leg-pain	9	33	9	38	0.762
	Syncope	16	59	9	38	0.126
	Fatigue	12	44	12	50	0.898
	Snoring	10	37	13	54	0.275
	Back pain	12	44	10	42	0.650
	Orthopaedic problems	11	41	15	63	0.125
Lifestyle factors	Current smoker	2[a]	7	2[b]	8	0.905
	Previous smoker	18	67	12	50	0.143
	Everyday Activity: Sedentary	9	33	5	21	0.830
	Light	10	37	12	50	
	Moderate	7	26	6	25	
	Vigorous	1	4	1	4	
Medication	Statins	23	85	21	88	0.815
	Antithrombotic	24	90	19	79	0.351
	ACEI	8	30	10	42	0.143
	Diuretics	7	26	10	42	0.242
	Calcium blockers	9	33	6	25	0.524
	Beta blockers	6	22	5	21	0.907
	Anticoagulants	2	7	3	13	0.551
	Other Antihypertensives	2	7	1	3	0.632
	Mean medication use	2.91 ± 1.09	2.85 ± 1.01		0.806	

Pack years. Note: Patients assessed for eligibility ($n = 167$) at the Hospital included the following demographics; age: 70 ± 13 years; sex: male $n = 93$ [56%], female $n = 74$ [44%]; Ethnicity: European $n = 137$ [82%], Maori $n = 12$ [7%], Pacifika $n = 9$ [5%], Asian $n = 6$ [4%], Indian $n = 3$ [2%].
[a]Current smokers who smoke between 0.5 and 1.0 pack per day.
[b]Current smokers who smoke <0.5 packs per day.

Vascular risk factors

Participants who took part in exercise and education intervention elicited a significantly greater change in SBP between baseline and postintervention than control ($P < 0.05$; $\eta_p^2 = 0.082$), with 61% (17 out of 28) of exercise and education intervention experiencing at least 10-mmHg reduction. The SBP values at 12PI were statistically similar to those reported at postintervention for both exercise and education intervention and control ($P > 0.05$; Table 3). Comparable findings were observed for bodyweight ($P < 0.05$; $\eta_p^2 = 0.061$) and BMI ($P < 0.05$; $\eta_p^2 = 0.070$), although the significant change in these measures was only

TABLE 3. Vascular risk factors reported for exercise and education intervention and control at each assessment (baseline, postintervention, 12PI)

	EX			CON		
	BL	PI	12PI	BL	PI	12PI
SBP (mmHg)[a,b]	140 (14)	129 (13)	129 (12)	139 (12)	138 (15)	138 (15)
DBP (mmHg)[b]	83 (9)	80 (9)	78 (9)	81 (9)	81 (10)	80 (11)
Pulse pressure (mmHg)[b,c]	57 (11)	49 (10)	51 (13)	58 (12)	57 (15)	58 (15)
Resting heart rate (b/min)	69 (14)	66 (12)	64 (9)	67 (14)	65 (8)	66 (9)
Double product (b/minmmHg)[b]	9.6 (2.3)	8.5 (2.1)	8.2 (1.2)	9.2 (2.2)	9.0 (1.7)	9.0 (1.7)
Total cholesterol (TC; mmol/l)[b]	4.02 (1.18)	3.46 (0.53)	3.54 (0.56)	3.93 (0.87)	3.89 (0.87)	3.83 (0.88)
High-density lipoprotein (HDL; mmol/l)	1.24 (0.54)	1.24 (0.58)	1.30 (0.54)	1.31 (0.52)	1.42 (0.49)	1.36 (0.46)
TC: HDL ratio[b]	3.77 (1.78)	3.27 (1.39)	3.13 (1.23)	3.28 (1.02)	2.97 (1.09)	3.02 (0.83)
Fasted blood glucose (mmol/l)	5.18 (1.54)	5.34 (1.34)	5.45 (1.22)	5.51 (1.09)	5.48 (1.14)	5.66 (1.03)
Bodyweight (kg)[a,b]	80.1 (16.5)	79.5 (16.0)	75.7 (16.0)	75.4 (14.7)	75.3 (14.5)	75.7 (14.0)
BMI (kg/m^2)[a]	28.8 (5.2)	28.5 (4.9)	28.2 (4.8)	28.0 (4.4)	28.0 (4.5)	28.2 (4.5)
Waist circumference (cm)	94.3 (14.7)	92.5 (13.4)	92.8 (12.2)	98.3 (13.5)	98.7 (14.2	96.1 (12.6)
Hip circumference (cm)	97.5 (11.4)	96.1 (11.9)	97.6 (11.8)	102.8 (13.1)	101.0 (13.4)	101.7 (14.7)
Waist: Hip ratio	0.97 (0.09)	0.96 (0.06)	0.95 (0.08)	0.96 (0.08)	0.98 (0.09)	0.95 (0.08)

Double product is a measure of the stress put on the cardiac muscle based on heart rate and the arterial blood pressure that it is pumping against. The value reported in the table is heart rate (b/min) multiplied by SBP (mmHg), divided by 1000. Values are reported as Mean (±SD). HDL, high-density lipoproteins; TC, total cholesterol.
[a]Significant condition by time interaction ($P < 0.05$); The observed change in SBP (between BL and PI) and for bodyweight and BMI (between PI and 12PI) for EX was significantly greater than CON.
[b]Significant time main effect ($P < 0.05$); Significant changes in outcome measures were observed between assessments (BL, PI, 12PI). As this analysis does not account for condition (EX, CON), the table infers that the observed changes across time points are largely attributed to those in the EX condition.
[c]Condition main effect ($P < 0.05$); A significant difference was observed in PP between groups (EX, CON) during the follow-up assessments (PI, 12PI).

observed between postintervention and 12PI (Table 3). Figure 2 demonstrates the change in SBP, bodyweight and BMI between baseline and postintervention, and baselien and 12PI. Similar trends were revealed for total cholesterol and resting double product ($P < 0.08$; Table 3). Despite similarities at baselien for pulse pressure, a lower value was reported for exercise and education intervention than for control during the postintervention and 12PI assessments ($P < 0.05$).

Aerobic fitness assessment

A greater oxygen uptake was reported for exercise and education intervention (33.7 ± 8.8 ml/kg/min) than for control (27.4 ± 8.8 ml/kg/min[1]) during the follow-up assessments ($P < 0.05$; $\eta_p^2 = 0.122$). On completion of the first stage of the cycle test (power output of 30 W; Table 4), a significantly greater change in SBP was observed between baseline and postintervention for exercise and education intervention than for control ($P < 0.05$; $\eta_p^2 = 0.103$). On completion of the second stage of the cycle test (power output of 60 W; Table 4), positive changes in SBP and DBP, pulse pressure and double product were observed between baseline and postintervention for exercise and education intervention (all $P < 0.05$). The improvements observed for exercise and education intervention were maintained at the 12-month assessment ($P > 0.05$; Table 4).

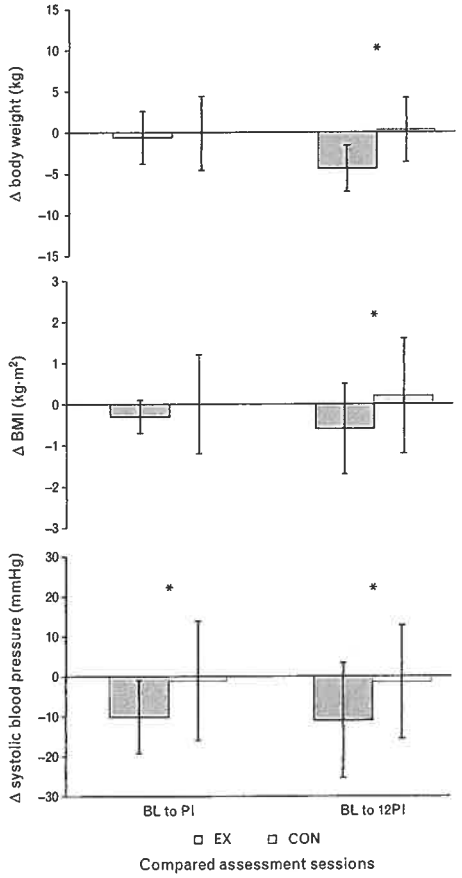

FIGURE 2 Change in SBP, BMI and bodyweight between baseline and postintervention and baseline and 12PI for exercise and education intervention and controls. Values are reported as mean (±SD).

DISCUSSION

This study examined the effects of an 8-week exercise intervention on the cardiovascular disease risk profile and aerobic fitness of newly diagnosed TIA patients, and whether these would be maintained at 12 months (12PI). We report that very early engagement in the intervention had a positive long-term effect (12PI) on resting and exercise markers of cardiovascular health (SBP, pulse pressure, double product, heart rate). The results are of interest for

TABLE 4. Physiological responses from the two stage cycle test for both conditions (exercise and education intervention, control), and for each assessment (baseline, postintervention, 12PI)

		Stage 1 (30 W)			Stage 2 (60 W)		
		BL	PI	12PI	BL	PI	12PI
Heart rate (b/min)	EX	94 (16)	91 (17)	92 (12)	107 (17)	103 (17)	105 (15)
	CON	96 (12)	96 (12)	97 (14)	110 (12)	113 (11)	115 (15)
SBP (mmHg)	EX	154 (18)	145 (19)[a]	144 (16)	173 (23)	158 (19)[b]	159 (19)
	CON	151 (16)	147 (16)	153 (14)	164 (17)	164 (17)	167 (17)
DBP (mmHg)	EX	85 (8)	80 (9)	79 (9)	87 (8)	81 (8)[b]	79 (7)
	CON	84 (9)	82 (11)	83 (11)	84 (11)	83 (11)	85 (11)
Pulse pressure (mmHg)	EX	66 (17)	65 (18)	65 (15)	87 (18)	76 (17)[b]	80 (18)
	CON	66 (15)	65 (15)	70 (14)	82 (16)	82 (19)	83 (18)
Double product (b/min*mmHg)	EX	13.9 (3.7)	13.0 (3.0)	13.0 (2.1)	18.7 (4.3)	16.1 (3.4)[b]	16.8 (3.4)
	CON	14.3 (2.7)	14.1 (2.8)	14.8 (2.8)	18.0 (3.3)	18.6 (3.4)	19.2 (3.9)

Double product. The value reported in the table for double product is heart rate (b/min) multiplied by SBP (mmHg), divided by 1000. Values are reported as Mean (±SD).
[a]Significant condition by test interaction between EX and CON during stage 1 ($P < 0.05$); The change in SBP between BL and PI is significantly greater for EX than CON.
[b]Significant condition by test interaction between EX and CON during stage 2 ($P < 0.05$); The change in SBP and DBP, pulse pressure and double product between BL and PI is significantly greater for EX than for CON.

three reasons: the study has 'real-world' validity because recruitment was conducted in accordance with normal health service provision for TIA, it reports on the longer term effects of an exercise and education intervention in TIA patients within 2 weeks of event, and the control group was aligned with standard care procedures in New Zealand, which are similar to TIA treatment recommendations in Europe and America [16,17].

On average, resting SBP, bodyweight and BMI were 7.9, 5.5 and 2.1% lower, respectively, for exercise and education intervention at 12PI than baseline. These changes were not observed for controls (Table 2), despite similar medication use between groups. When comparing groups at 12PI, resting SBP was 6.5% lower for exercise and education intervention than for controls. This is consistent with a meta-analysis that has demonstrated aerobic exercise training to elicit significant reductions in SBP [~4.7 mmHg; 95% confidence interval (CI) 4.4–5.0] and DBP (~3.1 mmHg; 95% CI 3.0–3.3) [18]. A lower SBP and DBP, pulse pressure, heart rate and double product was also observed for exercise and education intervention during the cycle test. The greater decrease in double product shown for exercise and education intervention, which was demonstrated during both follow-up assessments, demonstrates an important improvement in cardiac efficiency that may lead to improved skeletal muscle and cerebral perfusion.

As blood pressure is a powerful determinant of risk for stroke and intracranial haemorrhage [1], these 12-month findings are of clinical relevance, particularly when considering that past research has suggested that the benefits gained from regular physical fitness training do not persist following completion of an intervention [19]. Importantly, this study challenges the assumption that the effects of an exercise and educational programme are not sustained in TIA. Furthermore, there were no adverse cardiovascular or cerebrovascular events during the exercise intervention consistent with the Cochrane review on safety of exercise training for stroke survivors [20]. Although this is reassuring, the study was not powered to fully assess safety of early exercise intervention in TIA and further research would be required before the universal adoption of such programmes. In addition, as only 35% (60 out of 167) of the assessed TIA patients took part in the exercise programme, and as 42% of TIA patients ($n = 70$ out of 167 patients) were not eligible due to the inclusion/exclusion criteria, the widespread utility of such a secondary prevention programme for TIA patients requires further consideration. However, the study does report clinically important and sustained improvements in cardiovascular risk factors in a very high risk group of TIA patients. As the intervention was also a composite of both exercise and education, it is difficult to establish whether the observed changes were due to the exercise or education, or a composite of both. It is plausible that the changes observed at 12 months were more resultant on the education component rather than the 8-week exercise programme. Although the inclusion of a comprehensive educational component may be considered a useful adjunct to the exercise programme we do not currently know how valuable the implemented education programme was. In addition, the exercise intervention utilized both aerobic and resistance exercise. It is therefore not possible to determine whether the overall improvement in exercise and education intervention is a result of aerobic exercise, resistance exercise or a combination of both. Future research should focus on different types of physical activity interventions in order to determine the most efficacious form of exercise post-TIA diagnosis.

In conclusion, this study has demonstrated that the beneficial physiological effects of an early exercise and education programme post-TIA can be maintained for 12 months. As this study has 'real-world' validity and was conducted in conjunction with standard care procedures, the findings lend support for the development and implementation of lifestyle-modification programmes for certain TIA patient groups.

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None.

Conflicts of interest

None declared. AQ2

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Reviewers' Summary Evaluation

Reviewer 1

Strengths: Examination of the longer term impact of an exercise regime on vascular risk factors after transient ischaemic attack (TIA). Blinding of outcome assessors to patient group allocation.

Weaknesses: Lack of demographic detail on the source TIA cohort and thus the potential generalisability of the intervention is limited.

Uncited references

[21–23].